Ptosis, or blepharoptosis, is a decrease in the eyelid opening, or palpebral aperture. The condition can be either congenital or acquired. Ptosis typically refers to a lack of normal opening of the upper eyelid, resulting in its downward drooping. Less commonly, there can also be a lack of normal opening of the lower eyelid, resulting in its upward placement, referred to as inverse or reverse ptosis.

UNDERSTANDING PTOSIS
Acquired ptosis can be caused by a number of etiologies, ranging from those related to normal aging changes to potentially life-threatening conditions. Generally, ptosis can be filed into one of the following categories: aponeurotic, traumatic, mechanical, myogenic, or neurogenic (see Categories of Acquired Ptosis). Because ptosis can be a harbinger of a possible serious underlying neurologic condition (Table), it is imperative to be able to identify the slightest degree of ptosis, even in an asymptomatic individual, because the magnitude of ptosis does not indicate its etiology.

Eyelid Measurements
Although eyelid asymmetry is often an indicator of ptosis, it need not be present; ptosis can be unilateral or bilateral. Eyelid measurements (Figure 1) and assessment of eyelid function are imperative to accurately diagnose ptosis. These can easily be performed as part of a comprehensive eye examination. Typically, ptosis is identified by measuring palpebral apertures and/or marginal reflex distance-1 (MRD-1). Palpebral aperture is the distance from upper eyelid margin to lower eyelid margin. MRD-1 is the distance from the upper eyelid margin to the corneal light reflex produced from shining a light into the eyes. In order to ensure an accurate measurement for both of these tests, it is important to immobilize the frontalis muscle. To do so, place the palm of one hand on the patient’s forehead with a bit of downward pressure to stop him or her from raising the upper lids by use of the frontalis muscle (Figure 2). For both of these measurements, the patient should be looking straight ahead with no head or chin tilt.

Additional eyelid measurements include eyelid crease and levator function. The eyelid crease represents the insertion of the levator aponeurosis into the upper eyelid; it can be
measured as the distance from the
upper eyelid margin to the eyelid
crease when the patient is looking
down. Note that there can be more
than one eyelid crease on each side
and that disinsertion of the levator
aponeurosis results in a larger mea-
surement or greater distance from
the upper eyelid margin. Levator
disinsertion, or aponeurotic ptosis,
can occur as a result of normal aging
or from excessive eye rubbing or
tugging, as in repetitive rigid contact
lens removal.1,3

Levator function is defined as maxi-
imum excursion of the upper eyelid
from downgaze to upgaze and is
measured by having the patient look
down as far as possible and placing
the reference point of a ruler so that
it is contiguous with the upper eyelid
margin. Without moving the ruler
and without the patient moving his
or her head, have the patient look up
as far as possible. Where the upper
eyelid margin now intersects the ruler
(the distance the upper lid margin
traveled from downgaze to upgaze)
equals the levator function.1,3

**Eyelid Anatomy**

**Levator Palpebrae Superioris**

The levator palpebrae superioris is
the strongest of two eyelid muscles
that function to open the upper
eyelid. It is innervated by the third
cranial nerve. Therefore, damage to
the levator muscle itself, to its apo-
neurotic insertion into the upper
eyelid, to the third cranial nerve, or
to the neuromuscular junction between
the muscle and the nerve (as impaired
in myasthenia gravis) can each result
in a ptotic upper eyelid. Damage to
any of these structures related to
the levator palpebrae superioris may
result in any degree of ptosis, from
slight to complete eyelid closure.1,4

**Superior Tarsal Muscle**

The second eyelid muscle that nor-
mally functions to open the upper
eyelid is the superior tarsal muscle,
also known as the Müller muscle.
It originates from the undersurface
of the levator palpebrae superioris
and inserts into the superior tarsal
plate. The Müller muscle plays only a

**CATEGORIES OF ACQUIRED PTOSIS**

**APONEUROTIC** – (a.k.a. senile or involutional ptosis) The most common type of acquired ptosis related to aging. This includes disinsertion of the levator aponeurosis.

**MECHANICAL** – Occurs when the eyelid is too heavy for the muscles to keep it elevated due to reasons such as a mass or excess skin.

**MYOGENIC** – Unlike aponeurosis, caused by dysfunction of the levator muscle itself, which prohibits the eyelid from being elevated into proper position. This is less likely to be associated with damage to the Müller muscle.

**NEUROGENIC** – Occurs as a result of dysfunction or damage to the oculomotor or sympathetic nerves or to the central nervous system.

**TRAUMATIC** – Can encompass aponeurotic, mechanical, myogenic, and neurogenic ptosis that occurs as a result of traumatic damage.

Inferior Tarsal Muscle

Unlike the levator palpebrae superioris, the superior tarsal muscle has a companion muscle that acts to open the lower eyelid. The inferior tarsal muscle inserts into the inferior tarsal plate and is also under sympathetic autonomic innervation. Therefore, neurologic sympathetic autonomic damage, as in Horner syndrome, may result in both a small upper eyelid ptosis and a small lower eyelid reverse ptosis.1,4,5

Differential Diagnoses

Horner Syndrome

Because sympathetic autonomic innervation involves not only the tarsal muscles but also the iris dilator muscle, it is equally important to measure pupil size in both bright and dim illumination. A small degree of ptosis in the setting of anisocoria greatest in dim illumination, or even just a smaller pupil on the same side of the ptosis, should raise suspicion for Horner syndrome.

To prove that Horner syndrome is present in a nonacute and nonpainful setting, diagnostic testing with 0.5% or 1.0% apraclonidine is key. A reversal of anisocoria, in which the smaller pupil in the ptotic eye becomes the larger of the two pupils within 1 hour after instillation of the apraclonidine in both eyes, is diagnostic of Horner syndrome.

In cases of suspicious Horner syndrome that is acute and/or painful, diagnostic drops should be withheld and the patient should be sent immediately to the emergency department for neuroimaging (CT angiography or "MAKING EYELID ASSESSMENT A REGULAR PART OF COMPREHENSIVE EYE EXAMINATIONS CAN HAVE A POSITIVE EFFECT ON YOUR PATIENTS’ QUALITY OF LIFE ... .")
thenia gravis does not cause pupillary
illumination but also ductions and
measurements in both bright and dim
illuminations can also be clinical
features concerning for an aneurysmal
artery aneurysm. Aside from any
need for additional workup, urgency,
and potential treatment options.

Ptosis can be a harbinger of a serious underlying neurologic condition, such as Horner syndrome, third cranial nerve palsy, or myasthenia gravis.

Careful history-taking and past photo review, along with detailed measurements of efferent visual function, can help determine how to characterize a patient’s ptosis, the need for additional workup, urgency, and treatment.

MR angiography) to rule out carotid
dissection.1,6-8

Third Cranial Nerve Palsy
The other potential scenario requiring emergent neuroimaging in the setting of ptosis is a third cranial nerve palsy because it could be suggestive of a posterior communicating artery aneurysm. Aside from any degree of ptosis, other clinical features may include limitation of supra-, infra-, and adductions, with a corresponding reversing hyperdeviation and exodeviation that worsens when looking contralaterally to the side of the ptosis. In addition, because parasympathetic autonomic fibers travel on the outside of the third cranial nerve, a larger pupil on the side of the ptosis and/or anisocoria greater in bright illumination can also be clinical features concerning for an aneurysmal third cranial nerve palsy.9,10

Myasthenia Gravis
In the setting of a ptosis, it is critical to perform not only pupil size measurements in both bright and dim illumination but also ductions and cover testing in multiple positions of gaze. Another cause of ptosis combined with abnormal motilities is myasthenia gravis. Although myasthenia gravis does not cause pupillary abnormalities, it can cause other tell-tale clinical findings such as weakness of the orbicularis oculi muscle and positive results on fatigue and ice pack tests. Suspicion of myasthenia gravis usually requires outpatient workup and neurology referral, but it can be emergent in the setting of a myasthenic crisis.1,11-13

Pseudoptosis
When dealing with an eyelid asymmetry, it is also helpful to take exophthalmometry measurements because pseudoptosis can be caused by enophthalmos of the eye with the smaller palpebral aperture, or by proptosis and/or eyelid retraction of the fellow eye. Another common cause of pseudoptosis is dermatochalasis.1

TREATING PTOSIS
Careful history-taking and past photo review, along with detailed measurements of efferent visual function, as noted above, can help determine how to characterize ptosis, the need for additional workup, urgency, and potential treatment options.

After concerning underlying etiologies are excluded, the focus can be placed on treatment. Although surgery has been the mainstay of ptosis treatment to date, there is now a new option: preservative-free oxytetracycline HCl ophthalmic solution 0.1% (Upneeq, RVL Pharmaceuticals), which was approved by the FDA last year for the treatment of acquired blepharoptosis. This ophthalmic alpha-adrenergic agonist can be instilled once daily to raise the upper eyelid and improve superior visual field by activating the superior tarsal muscle.14

STAY ONE STEP AHEAD
Eye care providers should assess all patients for possible ptosis, even those who are asymptomatic. Making eyelid assessment a regular part of comprehensive eye examinations can have a positive effect on your patients’ quality of life in terms of morbidity and mortality as well as visual function and cosmos.


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